risk of injury. Thereby, the present finding could indicate that GJH may be an important risk factor for knee injuries and later development of OA.

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KNEE EXTENSOR MUSCLE WEAKNESS INCREASES THE RISK OF KNEE OSTEOARTHRITIS. A SYSTEMATIC REVIEW AND META-ANALYSIS

B.E. Øiestad †, C.B. Juhl ‡, I. Eizen §, J.B. Thorlund ‡. † *Norwegian research center for Active Rehabilitation (NAR), Oslo Univ. Hosp., Oslo, Norway;* ‡ *Res. Unit for Musculoskeletal Function and Physiotherapy, Univ. of Southern Denmark, Odense, Denmark;* § *Univ. Hosp. Oslo, Dept. of Orthopædics, Oslo, Norway*

Purpose: Previous studies including systematic reviews have suggested that knee extensor muscle weakness is a risk factor for the development of knee osteoarthritis (OA). However, systematic reviews have not aggregated the reported data into a meta-analysis. The aim of this study was to estimate the impact of knee extensor muscle weakness on the risk of knee OA.
Methods: A systematic review and meta-analysis was performed with literature searches in Medline, SportsDiscus, EMBASE, CINAHL, and AMED, all up to September 2013. Furthermore reference lists and systematic reviews on the topic were scrutinized for additional relevant studies. No restriction on language or publication year was made. Eligible studies had to include participants with no radiographic or symptomatic knee OA at baseline, and have a follow-up time of minimum 2 years. Studies had to include a measure of knee extensor muscle strength. Participants were classified as having knee extensor muscle weakness if they belonged to the lowest tertile or less in muscle strength or defined from a leg symmetry index (LSI). If more than one muscle strength outcome or knee OA definition were reported, a predefined hierarchy for extracting data was used. The hierarchy was: 1) Symptomatic radiographic knee OA for the whole knee, 2) Radiographic knee OA, and 3) Self-reported OA. Peak knee extensor muscle strength per kg body weight was preferred as measure of muscle strength; secondly average knee peak torque was extracted. Odds ratio (OR) for knee OA at follow-up was estimated in the included trials and combined using a random effects model. Stratified analysis for men and women were performed.
Results: A total of 1000 studies were identified thorough the literature searches. After review, four cohort studies with a follow-up time between 2.5 and 14 years were included. A total of 5102 participants (3325 men and 1777 women) were included in the final analysis. The studies included different groups of participants; middle-aged individuals with and without previous knee injury, elderly individuals without previous knee injury, as well as younger anterior cruciate ligament reconstructed (ACLR) individuals. Weak muscle strength was in 2 trials defined as being in the lowest tertile, when assessing the isokinetic knee extensor muscle strength. In one trial low muscle strength was defined as having a leg symmetry index of less than 80% (muscle strength in the affected leg was less than 80% of the muscle strength in the non-affected leg). Finally in one trial only data on the differences in muscle strength between patients with and without OA was available. Overall the meta-analysis showed an increased risk of knee OA in participants with knee extensor muscle weakness (OR 1.54 95%CI 1.28, 1.88; I^2 = 14.5%). Three trials reported separate data for men and women and subgroup analysis showed increased risk in men (OR 1.43 95%CI 1.14, 1.78; I^2 = 0%) and women (OR 1.79 95%CI 1.37, 2.31; I^2 = 3.3%), but differences in risk between men and women did not reach statistical significance (P=0.200).
Conclusions: Knee extensor weakness increased the risk of having knee OA at follow-up. Even though not statistically significant, the analysis indicated that weak knee extensor strength seems to be a stronger risk factor for OA in women than in men.

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RISK FACTORS FOR MAGNETIC RESONANCE IMAGING DIAGNOSED PATELLAR TENDINOPATHY IN COMMUNITY-BASED MIDDLE-AGED WOMEN

J. Toppi, J. Fairley, F. Cicuttini, J. Cook, S. Davis, R. Bell, F. Hanna, Y. Wang. *Monash Univ., Melbourne, Australia*

Purpose: Patellar tendinopathy, a condition that causes activity-related anterior knee pain, has diagnostic magnetic resonance imaging (MRI) features. The aim of this study was to examine the prevalence and risk factors for MRI-diagnosed patellar tendinopathy in community-based middle-aged women.
Methods: 176 women, aged 40-67 years, with no significant knee pain or injury underwent knee MRI. An MRI diagnosis of patellar tendinopathy was made if an area of increased signal intensity was seen in the proximal inferior patellar tendon region on at least 2 adjacent slices on both T1- and T2-weighted images. The cross-sectional area of vastus medialis was measured from MRI. Height and weight were measured to calculate body mass index (BMI). Physical activity was assessed using a questionnaire. Serum concentrations of C-reactive protein, androstenedione, dehydroepiandrosterone sulphate, testosterone, and sex hormone binding globulin (SHBG) were measured.
Results: The prevalence of MRI-diagnosed patellar tendinopathy was 30.1%. Higher levels of physical activity (odds ratio 1.65, 95% CI 1.09-2.51) and greater vastus medialis cross-sectional area (odds ratio 1.22, 95% CI 1.04-1.43) were associated with increased prevalence of patellar tendinopathy, independent of age and BMI. There were no associations with C-reactive protein, the sex steroids or SHBG.
Conclusion: In community-based middle-aged women MRI-diagnosed patellar tendinopathy is common, with higher levels of physical activity and greater vastus medialis size being risk factors. The findings highlight the importance of mechanical factors in the pathogenesis of patellar tendinopathy. Further work is needed to determine the contribution of patellar tendinopathy to knee pain and function.

OA: cartilage and bone

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PROGRANULIN A CHONDROPROTECTIVE GROWTH FACTOR IN THE PATHOGENESIS OF OSTEOARTHEITIS

B.S. Richbourgh, C. Liu, Y. Zhao, B. Liu. *NYU Sch. of Med., New York, NY, USA*

Purpose: The objective of this study is to determine the role of PGRN in human chondrocyte metabolism in vitro and in the course of OA progression in vivo, as well as the molecular mechanisms involved.
Method: Human chondrocytes and cartilage explants were isolated from OA patients. The isolated chondrocytes were cultured in the presence of TNF-α, PGRN, or both. Three days after treatment, cells and explants were collected and analyzed for anabolic and catabolic markers. Approaches for evaluating cartilage metabolism, including Safranin-O staining, immunohistochemistry, ELISA and Western blotting, were performed. A destabilization of medial meniscus (DMM) surgically induced murine model was generated with both wildtype and PGRN deficient mice. For testing the effect of recombinant PGRN in severe OA the ACL model was used. OA severity was evaluated using histological assay (e.g. Safranin-O staining), degradation of cartilage extracellular matrix molecules, and osteophyte formation.
Results: TNF-α dramatically reduced the levels of anabolic markers and enhanced the levels of catabolic markers including MMP13, ADAMTS-5, iNOS and COX-2, (Figure.1) while this alteration was largely abolished by additional treatment of PGRN, suggesting PGRNs protective role against human cartilage degeneration. Our results also demonstrated that PGRN greatly stimulates anabolic makers, including Aggrecan and Collagen type 2. (Figure.2). Histological analysis of cartilage isolated from DMM model revealed (Figure.3) significantly increased cartilage degradation in PRGN-/- mice relative to WT. Safranin O staining showed little or no proteoglycans in PRGN -/- mice along with significantly increased serum COMP fragment levels. Intra-articular injection of PRGN significantly reduced the degeneration of cartilage in surgically induced ACL model in WT mice, reflected by histological analysis of cartilage. In the ACL model 4 weeks-post surgery, PRGN treated mice retained cartilage integrity and showed little or no degradation of cartilage matrix in comparison to highly degraded cartilage of non-treated mice. Collectively, PRGN deficient mice develop more severe OA whereas treatment with PRGN markedly delays the progression of OA in WT mice.